ELECTROCARDIOGRAPHIC CHANGES DURING OPERATIONS FOR PULMONARY STENOSIS

BY

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When the operation of pulmonary valvotomy was started by Brock in 1948, one of the difficulties met was understanding the changes in the rhythm and action of the heart. It was thought that continuous electrocardiographic tracings might help in this way and so make it possible to lessen their incidence. Such tracings have generally been taken since then and in the present paper we wish to discuss the findings during 70 direct operations for pulmonary stenosis.

The patients fall into five groups: (I) those with Fallot's tetralogy (*a*) who had valvotomy for a valvular stenosis (13 cases); (*b*) who had infundibular resection (19 cases); (*c*) who had both these procedures (8 cases); and (II) those with pulmonary valvular stenosis with a closed ventricular septum (*a*) who were acyanotic (17 cases); and (*b*) who were cyanotic from a right-to-left interatrial shunt (13 cases). The records were analysed separately for these five groups, partly because the mortality was greater in groups I(*b*) and II(*b*). They were, however, very similar so are all discussed together though any special differences that were observed between the groups are commented on.

Similar records have been taken in many patients having mitral valvotomy and our findings (Campbell and Reynolds, 1952) will be compared. Although operations for pulmonary stenosis are, on the whole, more dangerous than those for mitral stenosis, probably because the surgical approach is through the right ventricle instead of through the left auricle, we find that the electrocardiographic changes are in some ways less than those during mitral valvotomy. The continuous tracings during angiocardiography (Reynolds, 1953) are also of interest for comparison.

Steps of the Operation

Anæsthesia has, in general, followed the lines laid down by Rink *et al.* (1948), and some aspects have been emphasized more recently (Rink, 1952; Hutton, 1952). After induction in the anæsthetic room, the patient is moved into the theatre and the first electrocardiogram is taken, usually 20–30 minutes after the induction. The area of incision is infiltrated with 1/400,000 adrenalin, the incision made, and the chest wall opened, using diathermy for hæmostasis. The left pleural cavity is then opened and the mediastinal pleura and thymus are dissected off the pericardium. Local anæsthetic is injected into the pericardial sac, originally 4 per cent procaine, but more recently 0·25 per cent decicaine.

After the nature of the lesion has been identified, procaine is injected into the space between the base of the aorta and pulmonary trunk, and then into the right ventricle at the point where the incision is to be made. At first, stay sutures or forks were inserted on either side of this area to control the bleeding after the incision was made, but now it is controlled entirely by digital pressure. In later cases pressures have been taken with an electrical manometer, either by a needle through the walls of the right ventricle and pulmonary trunk, or by means of a catheter after the incision into the right ventricle.

The obstruction is relieved by incision with a valvotome after exploration with a probe, and this is followed by dilatation with bougies and an expanding dilator: the number of times it is necessary to pass these instruments varies considerably in different cases. After recording pressures again, the incisions in the heart, pericardium, and chest wall are closed.

In the early cases when 4 per cent procaine was injected into the pericardium, the heart rate generally quickened from 10 to 30 beats a minute, probably from the local effect, but tended to slow when it was already above 125. In later cases, when decicaine was used, there was no significant change in the heart rate.

Changes of Supraventricular Rhythm

Nodal rhythm and changes of the pacemaker were often recorded in all the groups and at all stages of the operation (35 of 70 cases). They were not specially frequent at the time of valvotomy or infundibular resection and were seen more often later than this or at an early stage when the heart was first handled. They are probably not of much significance as they are not uncommon in patients with cyanotic heart disease at any time apart from operation, but they were much more frequent in those having infundibular resection (18 of 27) than in those having valvotomy only (17 of 43 cases). They were just as common in patients with mitral stenosis and normal rhythm during mitral valvotomy.

A-V Dissociation. A-V dissociation, often with interference, was found in at least six of these patients. In one where it was almost continuous through the whole record, the atrial and ventricular rates remained roughly parallel as though both were influenced in the same way by the same humoral or nervous factor. The atrial rate often became faster than the ventricular rate had been a few minutes before, but remained slower than the new ventricular rate. Over a period of about an hour the A-V ratio was between 100/101 and 100/115, generally between 100/105 and 100/110—A 134, V 142; A 125, V 132; A 130, V 140; A 135, V 148; A 128, V 139; A 132, V 145; A 130, V 145; A 128, V 148; A 137, V 148; A 146, V 150; A 136, V 137; A 134, V 137; A 133, V 135.

An example is shown in Fig. 1. It is unusual because each fifth ventricular beat is followed by

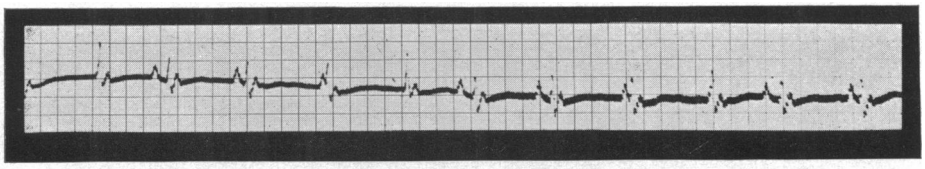

FIG. 1.—An unusual example of A-V dissociation with interference (see text). Case H168, lead II. Normal standardization was used for all the electrocardiograms.

a P wave that is itself early (A 62; V 65) for no apparent reason unless it is a slow response to the previous ventricular beat; the sequence is then repeated. Another example is shown in Fig. 3A. The sequence repeats itself after each four beats, the fourth ventricular complex being early, i.e. responding to the P wave of the previous beat with a P–R interval of 0·40 sec.: this is followed by a dropped beat and the ventricular rhythm is resumed.

Wolff-Parkinson-White Complexes. We have seen few instances of the Wolff-Parkinson-White syndrome in more than 1000 cases of congenital heart disease. The occurrence of such complexes in three patients during this operation is, therefore, of interest: we have not seen them in the corresponding series of mitral operations.

In two the picture was classical and three beats from one are shown in Fig. 2A. In the first they were observed between two occasions when the punch was used for infundibular resection but not just at that moment when it was being used. In the third (Fig. 2D) the earlier complexes might seem at first sight to be ordinary ventricular extrasystoles but measurement of the atrial and ventricular rates shows that in the first two the P wave, starting at just the same instant, is completely

hidden in the broadened ventricular complex; that in the third P keeps its place exactly but part of it can be seen with a P–R interval of 0·14 sec. before the ventricular complex because this is not quite so broad; and that in the fourth and fifth complexes P still keeps its place exactly but the P–R interval is lengthened to 0·16 sec. because the QRS complex is again a little narrower: his usual P–R interval was 0·20 sec.

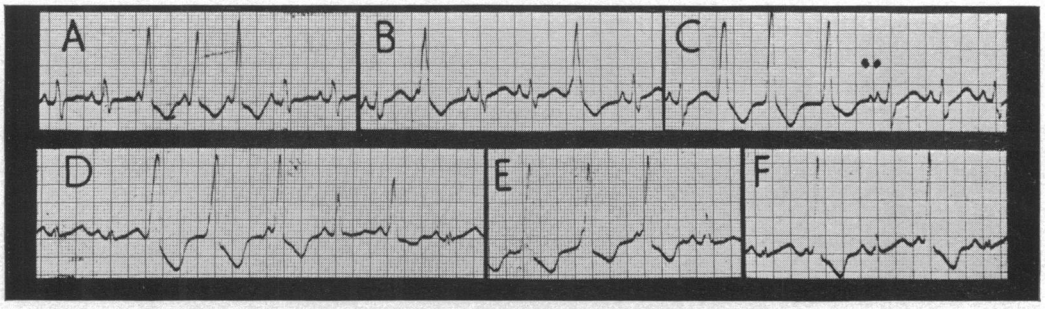

FIG. 2.—Examples of Wolff-Parkinson-White complexes. (A) Three classical complexes, (B) isolated complexes, and (C) less typical complexes followed by an unusual double P wave (see text). A, B, and C are from Case 0448, lead II; (D), (E), and (F) show less classical examples of the W.-P.-W. complexes (see text). Case 0263, lead III.

In the first patient there were also runs of what were apparently three ventricular extrasystoles (Fig. 2C), for though they were at a slow rate this was usual for him: we think these too may be similar to the records shown in Fig. 2D, and a further example of the W.-P.-W. syndrome.

Just after this another feature of interest is shown (Fig. 2C)—a double P wave. Partly from its appearance and partly because the whole record was very steady and free from extrinsic movements, this does not seem to be an artefact. We think that the first wave represents a left atrial complex but before this could initiate a ventricular complex it was blocked by a normal P wave arising from the right atrium in the sinu-atrial node, and this was followed by the normal ventricular complex.

VENTRICULAR EXTRASYSTOLES AND PAROXYSMAL VENTRICULAR TACHYCARDIA

Extrasystoles were nearly always ventricular. They occurred at any time but particularly when procaine was injected into the myocardium, when the sucker was used, and during the use of the valvotome. They were more frequent in the early stages of valvotomy or infundibular resection the first time an instrument was passed and often less frequent later, even when a larger instrument was passed.

In many patients only a few extrasystoles were recorded during the whole operation, perhaps no more than a dozen. When procaine was injected into the myocardium about one-third of the patients had one or two short runs of from three to five extrasystoles. Only twelve had more than this, the longest run consisting of ten consecutive beats, and the rest had fewer, sometimes much fewer than this. During the use of the valvotome there were 10 patients where the extrasystoles were frequent, and another 24 where they were fairly frequent, but in nearly half there were only a few extrasystoles even at this time.

There was no obvious difference between the different groups in the incidence of extrasystoles or of ventricular tachycardia: they were no more common with infundibular resection than with pulmonary valvotomy and no more common when the valvular stenosis was complicated by a right-to-left inter-atrial shunt. Both single extrasystoles and paroxysms of ventricular tachycardia were much less frequent, and when they did occur were generally shorter, than during operations for mitral stenosis, where they were a notable feature, especially at the time of mitral valvotomy. Ventricular extrasystoles are not uncommon in other operations and are most often due to a depressed pacemaker with local stimulation of the heart: they can also be caused by over-dosage with cyclopropane or trilene (Harris, 1953).

Paroxysmal Ventricular Tachycardia. In more than half the patients there were two or more paroxysms recorded at some time during the operation, and one-fifth had three or more recorded. Examples are shown in Fig. 3 and 4. Generally the paroxysms consisted of only a few consecutive extrasystoles. In 36 of the 70 patients the longest run consisted of five beats or less and in 16 of between six and nine beats. There were 12 patients where there were ten or more, but only three where there were more than fourteen beats. These three were a paroxysms of 37 beats during infundibular resection; one of 25 beats during valvotomy; and one of 17 beats when the catheter was introduced for pressure recording. The paroxysms of 10 beats were most often during valvotomy or infundibular resection but some followed the use of the catheter for recording pressure.

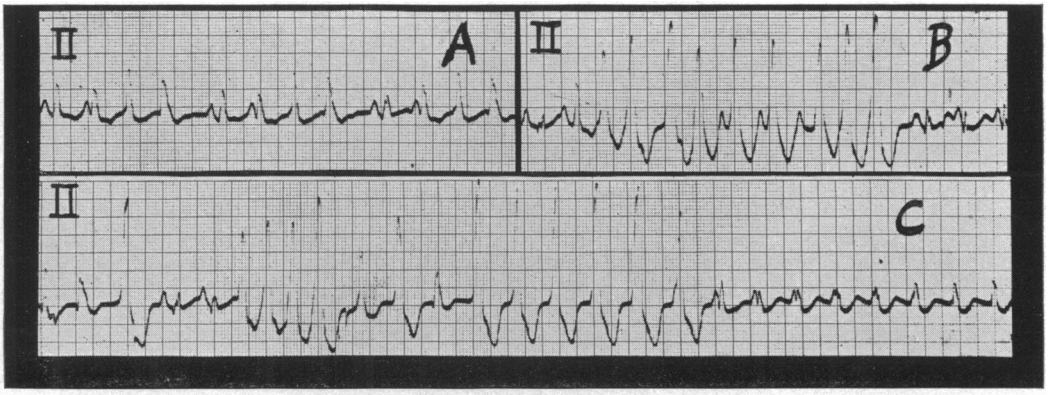

FIG. 3.—(A) A-V dissociation with interference (see text), (B) and (C) somewhat irregular paroxysms of ventricular tachycardia of 9 and 13 beats. Case 0700, lead II.

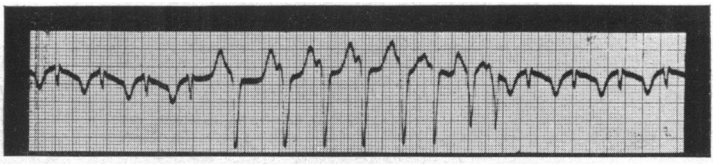

FIG. 4.—A short ventricular paroxysm that is almost regular in the middle period. Case 0089, lead II.

These short paroxysms differed from ordinary ones in being irregular, often considerably so. In one longer paroxysm the rate of individual beats varied between 114 and 206 but was generally not far from 124, as there were not many at the faster rates. Differences of this degree were quite common. There were, however, 54 paroxysms where by omitting a few of the less regular beats one could say that most of them did not vary greatly from the average rate.

In these 54 cases the rate was between 90 and 119 in one-tenth, between 120 and 179 in three-fifths, between 180 and 210 in one-fifth, and exceptionally fast, up to 260 in one-tenth. The unusually slow and the unusually fast rates could not be correlated with any special features of the patient or with any special stage of the operation.

Paroxysmal auricular tachycardia was rarely observed.

Generally, the paroxysms were as irregular as those during mitral valvotomy; they were not quite constant in form though less multifocal than those seen during mitral operations where predominantly R and predominantly S types and other striking changes of shape were frequent. Here nearly all the extrasystoles were of the R type and generally seemed to be produced by local stimulation at the base of the heart. We think that the obstruction to the blood flow by the introduction of the finger into the mitral valve explains the much greater frequency in mitral valvotomy.

Bundle Branch Block

Wide QRS complexes of the type seen in partial and complete right bundle branch block were common, being seen in more than three-fifths of the patients. They were often present for a large part of the operation, but had usually returned nearly, if not quite, to their normal width before it ended.

Generally the onset of such complexes took place within a short period but not absolutely suddenly, and the disappearance was even more gradual. For this and other reasons we think that many QRS complexes with a duration of more than 0·12 sec. are not true bundle branch block but are caused by a general delay of myocardial conduction from anoxia or other factors, and are more comparable with the complexes that may be seen with extreme ventricular hypertrophy.

There were, however, examples of sudden changes that seem to be true bundle branch block, e.g. Fig. 5A. It was not always easy to decide the side of the bundle branch block but we think that it changed from left to right in Fig. 9C, and in from right to left in Fig. 5A, for an S wave developed in lead III soon after the opening of the chest, presumably from rotation of the heart. The changes shown in the three leads were all recorded within a few minutes but, of course, were not simultaneous, so that there is no proof that we have placed them rightly.

The complexes nearly always had the form of right bundle branch block but they were of the left form in four patients in whom they did not then seem to have any different significance. The probable reason is simply that most of the patients already had right ventricular preponderance. Sometimes the width of QRS was already increased before operation and then a further increase even up to 0·12 sec. was only counted as partial bundle branch block.

One patient had complete right bundle branch block as her usual rhythm. For this reason and because she was 26 and had pulmonary stenosis with a right-to-left inter-atrial shunt her operation was undertaken with much anxiety. The changes were, however, minimal and everything went smoothly. She showed two extrasystoles only during valvotomy and three more when the pressure was being measured with a catheter: she showed no S–T depression at any time. During most of the operation she had nodal rhythm at a rate of 76, but sometimes there was A-V dissociation (see Fig. 1).

The frequency of bundle branch block was greatest in those with valvular stenosis and a right-to-left inter-atrial shunt and was similar in the other groups. It was more common (60%) than during mitral valvotomy (25%) and there, too, it generally had the form of right bundle branch block.

S–T Depression

The other important change that was seen frequently was S–T depression, often of considerable depth. It was observed at some stage in four-fifths of the patients and its incidence was similar in each group, except that it was less common in those with pure valvular stonosis. It often developed rather late, some time after valvotomy or infundibular resection. Sometimes it lasted for most of the operation and had not returned to the iso-electric level at the end, but more often it had nearly or quite disappeared before the operation finished. It was never present in records taken on subsequent days.

In about half the cases the depression was not great, being 3 mm. or less (13 cases, 1 mm.; 16 cases, 2 or 3 mm.). In the other half it was 4 mm. or more: it was about 5 mm. in 12, between 7 and 10 cm. in 9 cases, and in one, who caused no special anxiety during or after the operation, it was as much as 12 mm. Examples of fairly severe depression are shown in Fig. 6 from two patients shortly after valvotomy. The deeper depression was mostly in the patients who were having infundibular resection or in those with pure pulmonary stenosis and a right-to-left inter-atrial shunt.

Sometimes there was S–T depression in leads II and III with reciprocal elevation in lead I. When it followed the injection of local anæsthetic into the ventricular muscle at the site of the proposed incision it always had this form. At other times and especially during valvotomy and infundibular resection the depression was usually in all leads without reciprocal elevation. It was,

however, of the reciprocal form in 5 patients during valvotomy and in 5 others where it occurred later than this: two of these died (Cases 3 and 4) but the other three did well and caused no special anxiety.

It was seen more frequently in these operations (80%) than in those for mitral stenosis (62%) where it was of about the same degree. There, as during pulmonary valvotomy, it was not often of the reciprocal type.

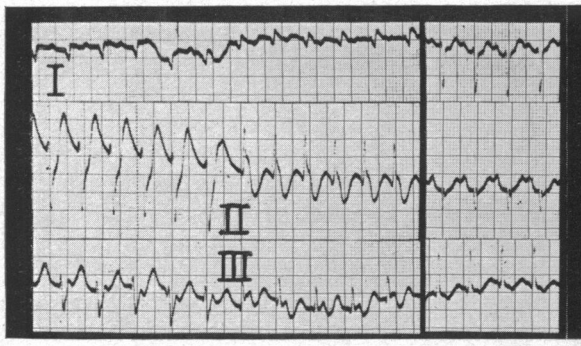

FIG. 5

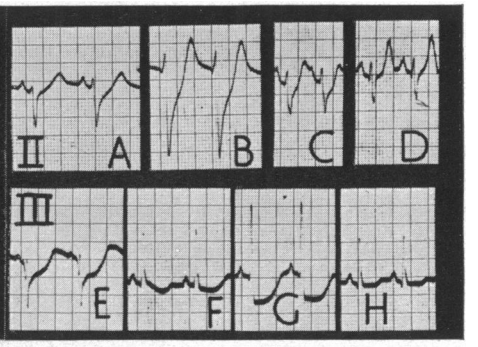

FIG. 6

FIG. 5.—Changes from right to left bundle branch block shown in all three leads within a few minutes, and on the right the disappearance of bundle branch block towards the end of the operation. Case P087, standard leads I, II, and III.

FIG. 6.—Severe S–T changes. (A) During collapse after valvotomy with some S–T depression. (B) Two minutes later, increasing S–T depression with nodal rhythm and bundle branch block. (C) Fourteen minutes later, deep S–T depression. (D) An hour later at the end of operation, the S–T depression has almost disappeared. (A)–(D) Case 0032, lead II. (E) Deep S–T depression sometime after valvotomy, probably due to the use of catheter for recording pressures. (F) Ten minutes later, almost normal. (G) Thirteen minutes later, severe S–T depression during closure of the heart. (H) An hour later at the end of operation with a return to normal. (E)–(H) Case 0024, lead III.

DIRECT VENTRICULAR TRACINGS

We have taken direct tracings from the surface of the ventricle before and after the injection of the local anæsthetic in seven cases. Generally about 3–5 ml. of 0·5 per cent procaine was injected at the site of the incision. There was always a large increase of S–T elevation, generally with a much smaller S wave (Fig. 8A). The increase of the S–T elevation in the electrogram was enormous, from 110 to 170 mm., in 4 of the 7 cases. Similar but slighter changes were seen for 1–2 cm. round the site of the injection (Fig. 8C), but not 3–5 cm. away from the site towards either border of the ventricular surface (Fig. 8B).

The degree to which these changes were shown in the standard leads varied and Fig. 7 gives an example where they were striking in the electrograms but much less in the standard leads; in spite of the magnitude of the changes they generally disappeared within a few minutes (Fig. 7C), probably as soon as the local anæsthetic was absorbed.

OTHER CHANGES OF RHYTHM

Heart Block. This was rare, apart from the examples of A-V dissociation already described, and even lengthening of the P–R interval was uncommon. It may, of course, have been missed when tachycardia led to the P wave being hidden in the preceding T wave, but we do not think this happened often. There were, however, three cases where heart block developed. In one who recovered, there was 2:1 heart block and dropped beats; the other two died, Case 4 showing 3:2 and 2:1 heart block and Case 5 showing partial heart block with dropped beats.

Auricular Fibrillation and Flutter. No example of either of these rhythms was observed. This agrees with clinical experience that they are uncommon in the types of congenital heart disease in question.

Ventricular Fibrillation. This was observed in one patient who lived for ten days afterwards (Case 1, see below), and in two who died. In the first it followed a period of irregular ventricular rhythm and cardiac standstill. In the second (Case 2) it occurred before valvotomy had been attempted and followed a period of cardiac standstill. In the third (Case 3) it followed ventricular tachycardia: after a short period sinus rhythm returned but only for a time.

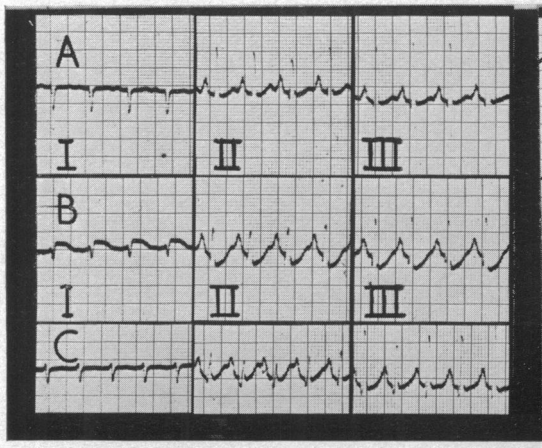

FIG. 7

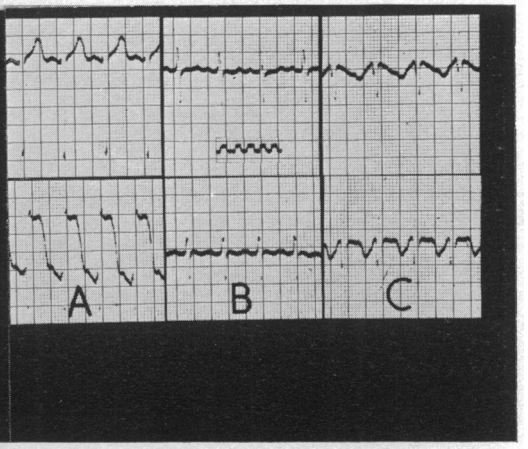

FIG. 8

FIG. 7.—(A) Before, (B) immediately after, and (C) some minutes after injection of procaine into the myocardium. (B) Shows the amount of S–T elevation and depression that is reflected in the standard leads. Case OA04, leads I, II, and III.

FIG. 8.—Electrograms from the surface of the ventricle before and after the injection of procaine. Above, before injection; below, after injection. (A) Gross S–T elevation, at the site of the injection. (B) Little or no S–T elevation, at the antero-lateral border of the right ventricle. (C) Slight S–T elevation, 1–2 cm. away from the site of injection. Compare Fig. 7 which shows how far these changes were reflected in the standard leads. Case OA04. The standardization of all these was greatly reduced.

TEMPORARY RECOVERY AFTER VENTRICULAR FIBRILLATION

Case 1 (0133). A boy, aged 7, with Fallot's tetralogy. There was, as often, some temporary flattening of the T waves when procaine was injected into the heart, and some widening and notching of the QRS complexes. During infundibular resection there was complete bundle branch block and soon after paroxysmal ventricular tachycardia of the S type that did not seem to have been caused by any particular use of the finger or punch. It was difficult to say exactly when the paroxysms started and ended as the rate increased and decreased less suddenly than is usual (Fig. 9B).

Later there was A-V dissociation and a change from left to right bundle branch block (Fig. 9C). A moment later there was a period of slower and irregular ventricular rhythm with asystole for four seconds after which there was ventricular flutter. This lasted about four minutes and included a very short period of more irregular action like ventricular fibrillation (Fig. 9D). He then resumed a slow ventricular or nodal rhythm and this was again interrupted by asystole for six seconds, which was not followed by ventricular flutter on this occasion. The ventricular rhythm gradually became faster and this stage lasted for about 45 minutes till he left the theatre. Half an hour later in the ward the cardiogram was normal except for slightly wide QRS complexes and in another half an hour this had passed off (Fig. 9E). The patient died ten days later but it seems that the heart itself had recovered, probably permanently, and that he died because of the cerebral damage sustained when he was without any effective cerebral circulation.

COURSE OF EVENTS IN PATIENTS WHO DIED

Four of the 70 patients died during the operation. One had pulmonary valvular stenosis with a right-to-left inter-atrial shunt and the others had Fallot's tetralogy. They collapsed at very

different stages—Case 2 before the operation had been started, Case 3 just after the finger was inserted in the infundibulum, Case 5 soon after the completion of the valvotomy and resection, and Case 1 who recovered temporarily, some time after the infundibular resection; Case 4 seemed all right till the chest was being closed nearly an hour after the infundibular resection.

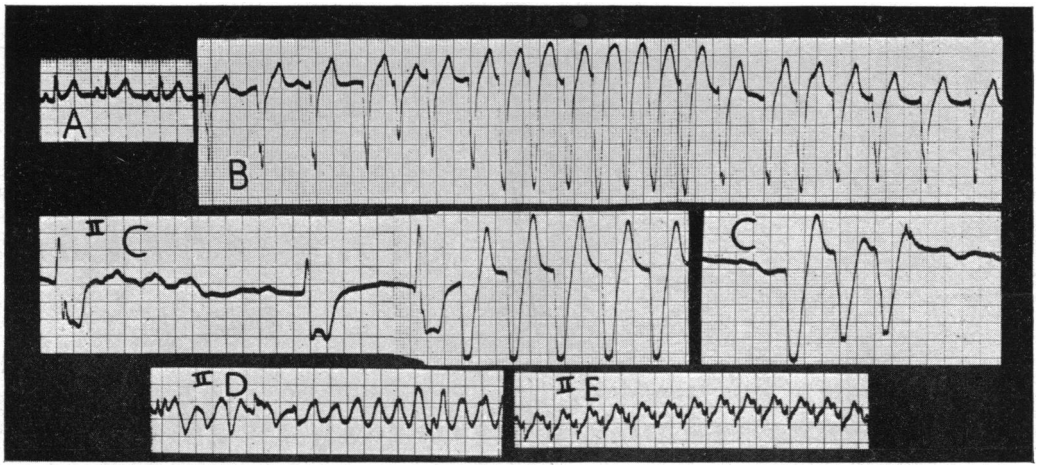

FIG. 9.—Ventricular tachycardia (B), bradycardia, and ventricular fibrillation (D), in a patient who made an immediate recovery but died ten days later. See text. Case 1 (0133), lead II.

Sometimes the electrocardiogram gave the earliest warning that the patient's condition was becoming serious (e.g. Case 3): sometimes the patient collapsed with a relatively normal cardiogram and changes in this came later (Cases 4 and 5). These four patients are described more fully.

Case 2 (0041). This girl, aged 10, had pulmonary valvular stenosis with a closed ventricular septum and a right-to-left inter-atrial shunt. She had deteriorated greatly during the year before operation and the heart had become larger.

The first cardiogram, taken with the patient on the operating table but before the operation had started, was already abnormal, showing a nodal rhythm, 60 beats a minute, and 2:1 retrograde block (Fig. 10A). The rate soon slowed so that four minutes later it was only 37 and the QRS complexes widened to 0·15 sec. A moment later the heart appeared clinically to have stopped completely. An intra-cardiac injection of adrenalin was given and the chest was rapidly opened. The rate continued to fall until four or five minutes after the incision it was 22: the QRS width was now 0·26 sec. and there was no certain evidence of auricular activity (Fig. 10B).

The QRS then almost divided into two separate complexes with a total width of 0·52 sec. (Fig. 10C) and a minute later the rhythm became irregular with rates up to 95 a minute (Fig. 10D), though a regular rhythm returned from time to time. Intracardiac adrenalin was again given and a minute later there were two short runs of ventricular tachycardia at rates of 175 and 165 (Fig. 10E), after which bradycardia (35 a minute) appeared again with asystole for 3 seconds.

The pericardium was now opened and the forks to control bleeding from the incision in the right ventricle were inserted. Ventricular flutter at 270 a minute appeared and was at once followed by fibrillation (Fig. 10F). This alternated with multifocal ventricular tachycardia for the next 15 minutes, in spite of an intracardiac injection of procaine. The ventricular rhythm persisted (Fig. 10G) and soon slowed to 60 a minute, perhaps as the result of an injection of calcium chloride. The rate rose again to 120 and continued to vary a good deal and finally slowed (Fig. 10H) to complete asystole about 40 minutes after the onset of ventricular fibrillation and an hour after the first cardiogram.

Case 3 (P063). This man, aged 28, had Fallot's tetralogy, and infundibular resection was

carried out as he was becoming progressively disabled. There was some S–T depression soon after the chest was opened, and three ventricular extrasystoles when procaine was injected, but no noticeable effect when the incision into the heart was made. Occasional single extrasystoles followed the use of the valvotome but up to this time the record had shown very little disturbance.

Immediately after the finger was used in the infundibular stenosis, QRS became wider and there were more ventricular extrasystoles and S–T depression, and a minute later frequent multifocal ventricular extrasystoles (Fig. 11A). After another half minute, there was ventricular fibrillation

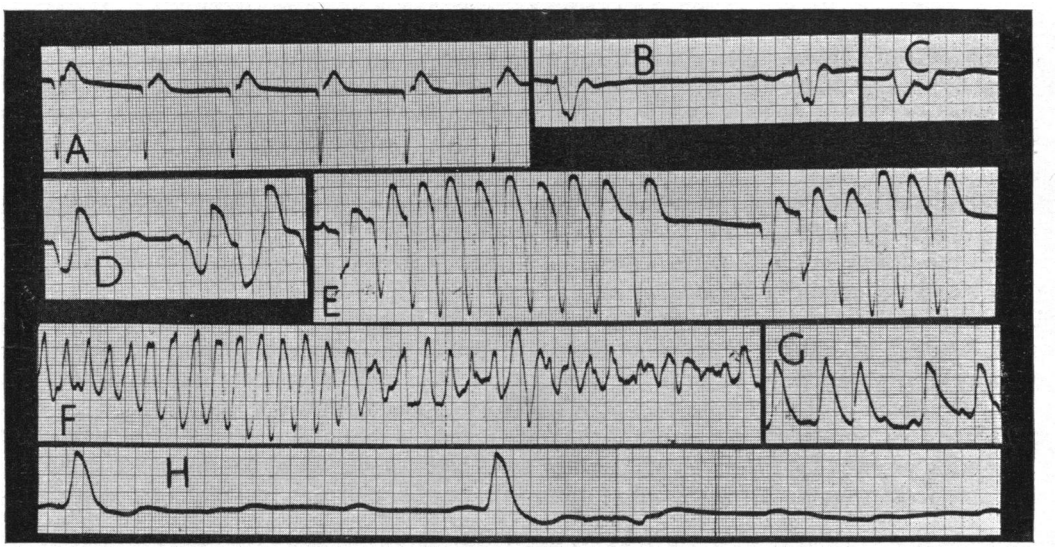

FIG. 10.—Slow nodal rhythm, paroxysmal ventricular tachycardia (E), ventricular flutter and fibrillation (F), followed by a very slow ventricular rhythm and some signs of auricular activity (H) as the patient was dying. See text. Case 2 (0041), lead I.

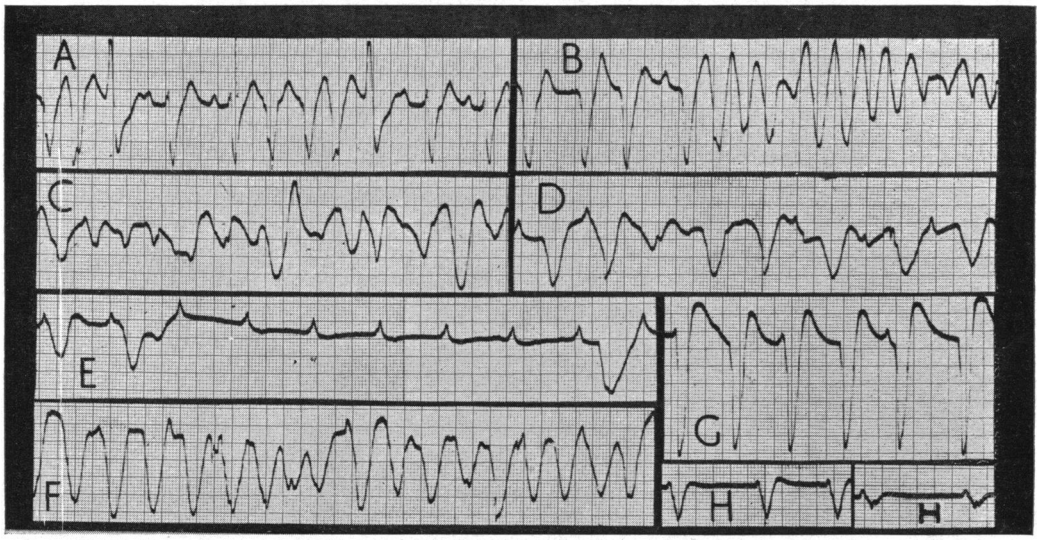

FIG. 11.—Multifocal ventricular extrasystoles (A), ventricular flutter (B), and fibrillation (C and F) with a slow ventricular rhythm (H) as the patient was dying. See text. Case 3 (P063). (A) lead III, (B) lead II, rest II or III.

and short runs of ventricular flutter (Fig. 11B), followed by a type of fibrillation that was coarser than usual (Fig. 11C).

Ventricular complexes then became wider at a rate of 100 and the P waves could now be seen clearly at a rate of 75 (Fig. 11D). There was a period of asystole lasting 5 seconds with regular P waves at a rate of 77 (Fig. 11E) and then a more rapid ventricular fibrillation, still with visible P waves (Fig. 11F). After 8 minutes of this there was a slower ventricular rhythm (Fig. 11G). By this time the patient was thought to have died but attempts at resuscitation were continued for another 15 minutes. The slow ventricular rhythm continued for half an hour, the voltage gradually getting smaller and smaller (Fig. 11H).

Case 4 (0224). This woman, aged 19, had Fallot's tetralogy. When the anæsthetic was started the T waves became flatter as often, and when procaine was injected into the heart there were a few extrasystoles and nodal escape. When the incision into the ventricle was made there was a single ventricular extrasystole, and when a probe was passed through the valve there were two extrasystoles and rather more when the punch was used, all these being of the R type (Fig. 12A). A few minutes later with the use of dilating forceps, there was temporary S–T depression and when the finger was used this increased and there were also extrasystoles (Fig. 12B); but neither lasted long, and when the infundibular resection was completed the electrocardiogram was practically normal (Fig. 12C) and her condition seemed good.

Half an hour later as the chest was being closed there was some S–T depression, but this is not unusual. Nearly an hour after the valvotomy Mr. Brock was not satisfied with the patient's condition but the electrocardiogram was quite unchanged. A minute later there was sinus bradycardia at about 60 instead of the previous rate of 130. It quickened to 130 but slowed to about 46, with nodal escape and then A-V dissociation with interference (A about 40 and V about 48); the patient was becoming worse. The T waves were rather more biphasic but there was not much else to show. Fourteen minutes after the patient's deterioration the cardiogram was really abnormal for the first

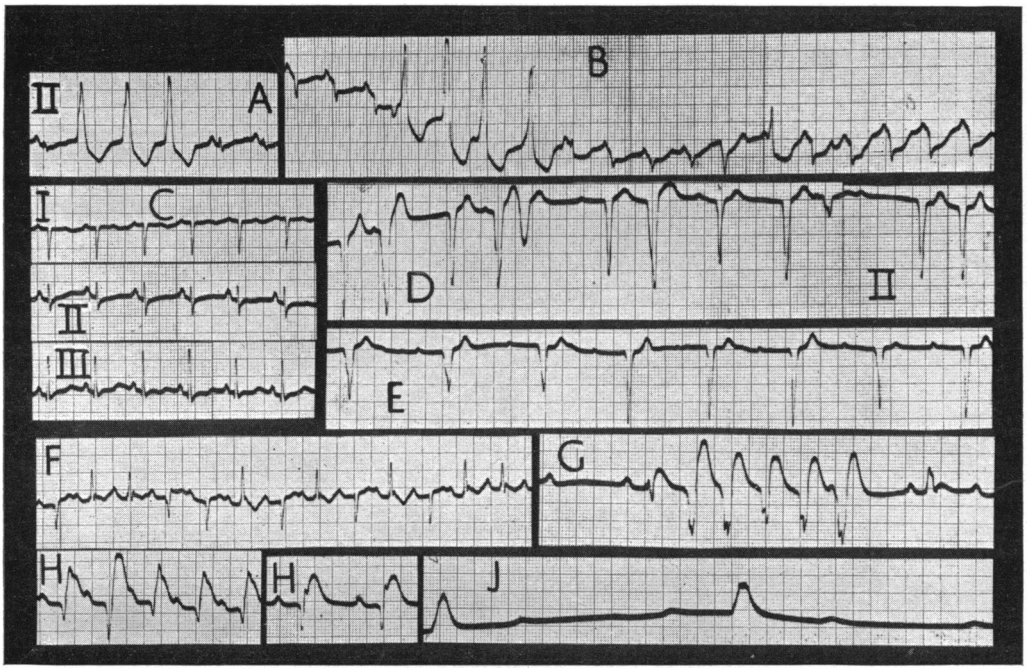

FIG. 12.—Ventricular tachycardia, irregular ventricular rhythm (D), bidirectional ventricular complexes (F), ventricular tachycardia (G), and gross S–T elevation (H), with final and slow ventricular rhythm with some evidence of auricular activity (J) as the patient was dying. See text. Case 4 (0224), lead III, if not marked.

time with large and more irregular ventricular complexes for a few seconds (Fig. 12D). The P–R interval then lengthened to 0·42 sec. with similar large ventricular complexes (Fig. 12E). There was then a slow sinus rhythm and escape, the QRS complexes widening up to 0·12 sec., and occasional runs of three or four ventricular extrasystoles. There was some anxiety about the patient's condition but it did not seem very grave. A few minutes later there was a good response to methedrine and the cardiogram became normal at 122 with a P–R interval of 0·18 sec. However, it gradually slowed again to 75 and two different types of ventricular response appeared. The P–R interval gradually lengthened to 0·34 sec. with dropped beats and sometimes with ventricular escape. After a further recovery and relapse there was a short period with bidirectional ventricular responses (Fig. 12F), then 2:1 heart block with widened QRS complexes, then complete dissociation (A, 106; V, 61), then 2:1 heart block with a P–R interval of 0·44 sec., and later 3:2 heart block. The patient's condition was getting worse and 36 minutes after her collapse there was a regular ventricular tachycardia at about 140 (Fig. 12G). This was followed by a very abnormal S–T elevation of two types (Fig. 12H). The auricular rhythm then became slow (52) and irregular with some ventricular responses, the P–R interval increasing from 0·28 and 0·63 sec. The auricular rate fell further and in the final records, 48 minutes after her collapse, there was A-V dissociation with the atrial rate about 34 and the ventricular rate about 17 (Fig. 12J).

Case 5 (0057). A girl, aged 5, with Fallot's tetralogy complicated by a single A-V valve. In the early stages of the operation there was no more than partial bundle branch block and after the injection of procaine a few ventricular extrasystoles followed by some temporary S–T depression. The valvotomy itself was uneventful: the S–T depression had gone and there were only a few ventricular extrasystoles, the longest run being three beats. The operation seemed shorter and less traumatic than many.

There was no clinical or cardiographic anxiety until half a minute after the punch had been used for the last time, when about the same time her condition deteriorated clinically and partial heart block with dropped beats was seen for 20 seconds. It seemed that the collapse was peripheral rather than directly cardiac. A minute later there were for the first time multifocal extrasystoles (Fig. 13A) and later the rhythm slowed from 120 with a long P–R interval to a sinus bradycardia (Fig. 13B). This has generally been a most unfavourable sign.

Within a few minutes she seemed to have died, though attempts at resuscitation were continued for more than half an hour. Next there was a sinus tachycardia with a long P–R interval, then multifocal extrasystoles and then bradycardia again for 5 minutes, when cardiac massage was used. This was followed by nodal rhythm with an inverted P and a shorter P–R interval and then by alternating slower sinus (61 to 55) and nodal rhythms (65 to 58) with multifocal extrasystoles (Fig. 13C). After this there were again asystole for 6 seconds, and then sinus rhythm with P waves varying from 105 to 48 with a 2:1 response; this went on for about 6 minutes and was followed by about the same period of a slow irregular ventricular rhythm (Fig. 13D).

The rhythm then changed to ventricular flutter at a rate of 150 (Fig. 13E), becoming slower and irregular, ventricular fibrillation (Fig. 13F) and then regular again at 120. These changes recurred two or three times and there was a regular rate as fast as 165 and a slow and irregular ventricular rhythm with probable evidence of auricular activity at about 100 (Fig. 13G). At one time this was more obvious and regular at 108 with A-V dissociation. The last records showed ventricular beats only, not very irregular, at about 12 a minute.

Discussion

The body of this paper has been a description of the course of events rather than a discussion of the causes: it shows the difficulties inherent in the method of clinical observation as opposed to that of experiment. The many facets of anæsthetic and surgical technique must be decided by the surgeon and anæsthetist as seems best for the patient, and single factors cannot be changed as they can be in experiment, to judge their importance.

Cardiac arrhythmias are specially liable to occur in intrathoracic surgery and it is known that

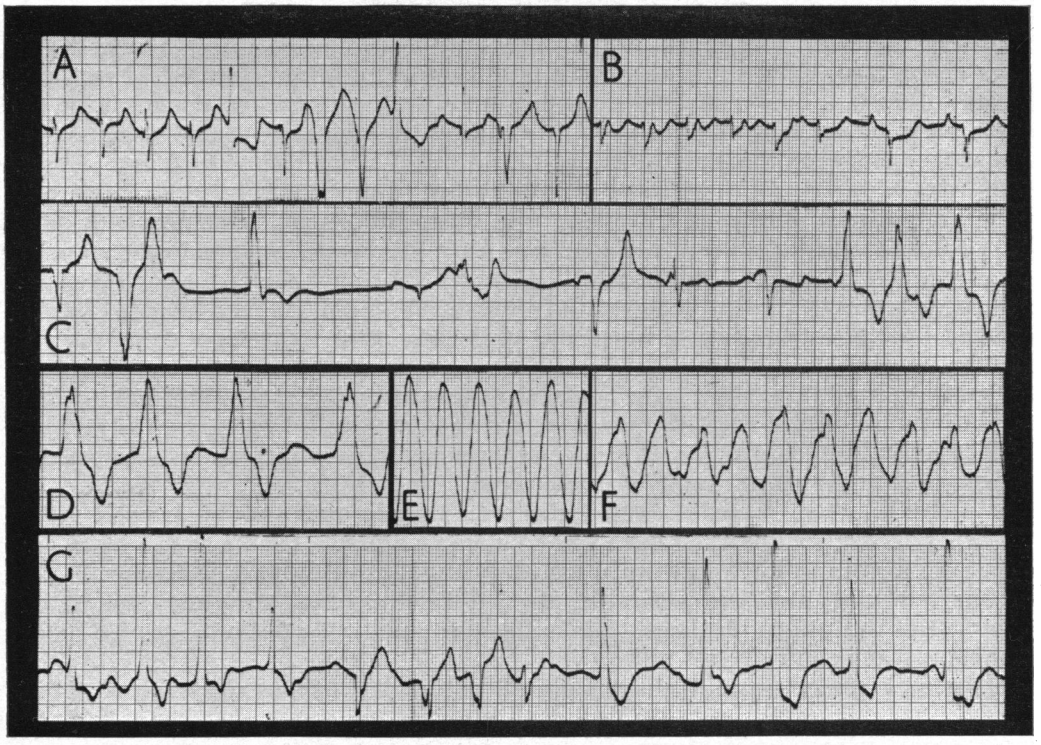

FIG. 13.—Bradycardia with multifocal extrasystoles (C), ventricular flutter (E), and fibrillation (F) with a return of auricular and ventricular rhythm (G) but without clinical recovery. See text. Case 5 (0057) (B) lead II, all others lead III.

cyclopropane may cause ventricular tachycardia or even fibrillation since Meek *et al.* (1937) showed this in dogs. It may, therefore, be felt that cyclopropane is an unwise choice for cardiac surgery, but the anæsthetists concerned think it has proved its value by the good results obtained in so many patients with grave heart disease.

Often it is not clear how far electrocardiographic changes recorded during the operation are due to the anæsthetic and how far to the operation itself, though the observations of Ziegler (1948) on similar patients having subclavian-pulmonary anastomosis suggest that the changes found much more frequently in our series are due to the direct operation on the heart. For example in his series, heart block and complete bundle branch block were found only in those who died and this was also true of the only three examples of paroxysmal ventricular tachycardia and the only example of ventricular fibrillation.

Anæsthesia is unfamiliar ground to most cardiologists; and anæsthetists, who have so much empirical and almost instinctive wisdom, are often without an equal knowledge of the changes in the heart and its rhythms. Although continuous electrocardiography during anæsthesia has been used for some years and discussed in a series of papers by Johnstone (1950–1953) it is still a new subject with much to be learned. Two conclusions can, however, be reached with certainty.

The short paroxysms of ventricular tachycardia that accompany mitral and, less constantly, pulmonary valvotomy, recur with the regularity of experimental results and here one can be certain of cause and effect. Their greater frequency during mitral valvotomy, especially at the moment when the finger is inserted in the valve suggests that obstruction of the circulation is the main cause, though direct trauma must also be considered.

The S–T depression and reciprocal elevation that follow the injection of local anæsthetic into

the ventricle are equally clearly caused by this injection and disappear quickly as it is absorbed. The cause of the S–T depression at other stages is less obvious. It is uncommon (4%) in operations for subclavian-pulmonary anastomosis (Ziegler, 1948) and is rarely seen in ordinary operations (Johnstone, 1953*b*) though Jaruszewski *et al.* (1953) have noted it and think it is due to anoxia. Anoxia may be the explanation of some of our cases though direct trauma to the heart seems the more likely reason for its frequency. Harris (1953) points out that S–T depression and bundle branch block may be produced by over-dosage with cyclopropane, trilene, and intravenous procaine as well as by anoxia.

Bundle branch block during anæsthesia will not be detected without electrocardiograms, but even with these Johnstone (1953*a*) finds it rare. Reporting four examples in patients with normal hearts he points out that many other hearts had been brought to the stage of standstill from vagal stimulation without it appearing. In his cases it followed the inhalation of ether when atropine had been used in relatively small doses, so was probably caused by pulmo-cardiac reflexes. He also discusses the difficulty of distinguishing bundle branch block and ventricular rhythms on single records—a point of practical importance because, in his experience, ventricular rhythms can generally be stopped by stimulating the vagal pulmo-cardiac reflexes and so abolishing the ventricular irritability due to sympathetic over-activity.

The many changes in the pacemaker and the frequency of nodal rhythm and of A-V dissociation with or without interference are almost certainly due to the anæsthetic. They are common features of anæsthesia in many operations (Kurtz *et al.*, 1936) and were frequently found by Ziegler (1948) at all stages of anæsthesia, and by Jaruszewski *et al.* (1953) especially before the operation started and during manipulations of the pleura and mediastinum. A-V dissociation is uncommon in extra-pulmonary operations except with too light anæsthesia or after over-dosage with cyclopropane or trilene (Harris, 1953). Generally, these changes in the rhythm are almost certainly caused by reflex vagal inhibitions: the vagus influences the S-A node more than the A-V node, so that the normal pacemaker easily loses control. In themselves they are probably without significance except as a warning that a higher degree of vagal inhibition may follow.

Sinus Bradycardia and A-V Block. It is clear from these cases that undue slowing of the heart should give rise to much more anxiety than tachycardia. Harris (1951) has stressed the anxiety that should be felt in general anæsthesia when the heart rate becomes unduly slow. He states: " . . . Vagal over-action during the stage of anæsthetic sleep may slow the pulse and may even produce cardiac arrest." But he also states: " A rise of pulse rate produced by anoxia or by a deficient venous return is of serious import and unless relieved, tachycardia of this origin may result in cardiac arrest." These two contrasted states of bradycardia and tachycardia, where both may lead to a serious issue, will be referred to again in discussing the patients who have died.

It is well recognized that vagal reflexes can go further and produce A-V block when, unless the patient is to die, the ventricle must start its independent rhythm. These changes can, of course, be caused by anoxia and other factors, including direct surgical intervention, but when, as in some of these cases, they follow on sinus bradycardia, it seems likely that they are largely a vagal effect and may perhaps be prevented by more atropine.

Ventricular Rhythms. The relationship between the different ventricular rhythms is not well understood. Normally, the independent ventricular rate is 25 to 40, but in younger patients it may be 50 or 60, and in conditions such as diphtheria, and perhaps anæsthesia, even faster. In these cases, as might be expected, the ventricular rhythms show many abnormal features. The slower rhythms are generally irregular and often vary in form in a way that is unusual in complete heart block. It is uncertain whether there is any fundamental difference between the faster and the slower paroxysms of ventricular tachycardia, and certainly in these records during operation it seems hard to draw any dividing line. At the upper extreme the rates have been as fast as 200 to 210 and occasionally about 250. Lucas and Short (1952) have failed to find any evidence that procaine amide helps in the *prevention* of these arrhythmias, though its success in terminating them seems that its use should be considered. It is fairly certain that at these rapid rates there is no

adequate mechanical contraction of the heart and that the output is little or no better than with ventricular standstill.

This is certainly true when the next step of ventricular flutter is reached. The existence of this rhythm has been doubted but very characteristic curves are shown in many of these patients (Fig. 10F, 11B, 13E, and perhaps 9D), curves that have the features of a continuous wave similar to the picture of auricular flutter. It is not very dissimilar from some paroxysms of ventricular tachycardia, but seems to be an entity. Although fairly common in these records during operation, it is rarely seen in ordinary conditions. A further reason for thinking that it is a real entity is the way in which it is so constantly followed by the much more irregular tracings that are accepted as ventricular fibrillation.

Ventricular Fibrillation and other Cardiac Rhythms at the time of Death. There is so much individual variation that it is not easy to generalize but certain sequences seem of special significance. In this series there were five deaths—four during the operation and one ten days later from the cerebral damage that occurred at operation (Case 1).

Case 1 showed a slow ventricular rhythm, asystole, ventricular flutter and fibrillation, followed by good recovery of the heart. Case 2 died with slowing of the heart followed by cardiac standstill: ventricular flutter and fibrillation were recorded only *after* the patient was thought to be dying. Case 3 had multifocal ventricular tachycardia followed by ventricular flutter and fibrillation. Case 4 had severe sinus bradycardia and about 14 minutes later bidirectional QRS complexes, ventricular tachycardia, A-V block, and finally a slow ventricular rhythm. Case 5 had severe sinus bradycardia at the time she was thought to be dying but multifocal ventricular rhythm and ventricular flutter were recorded while resuscitation was being attempted.

In general, therefore, sinus bradycardia was a grave sign (Cases 1, 2, 4, and 5); in Case 3, however, ventricular tachycardia was the first serious sign and was quickly followed by ventricular flutter and fibrillation. 2:1 complete heart block only occurred in three patients and two of these died, so that it seemed nearly as dangerous a sign as ventricular flutter and fibrillation.

The findings during mitral valvotomy and during angiocardiography will be briefly compared with these.

During Mitral Valvotomy. Four of these patients died from direct cardiac causes during operation, though in two of these the heart had recovered for a time and death followed the cerebral damage sustained while the circulation was ineffective. Three of these four had ventricular flutter and fibrillation (Campbell and Reynolds, 1952).

The first (G7) had complete A-V block, which disappeared when cyclopropane was stopped, before the chest was opened. Afterwards there was bundle branch block and S–T depression, multifocal ventricular tachycardia, and then asystole for two seconds. The ventricular rhythm became faster and changed to ventricular flutter at 150 and fibrillation, which lasted for 7 minutes. Normal rhythm was restored after methedrine but the patient did not recover consciousness and died four hours later.

The second (G15) was rather similar. The P–R interval lengthened to 0·34 and during valvotomy to 0·60 sec., and QRS widened to 0·18 sec. There was then 2:1 and complete A-V block with a slow ventricular rhythm which gradually quickened and changed its form to ventricular flutter and fibrillation; this was before the injection of adrenalin though fibrillation recurred after its use. Normal rhythm returned but she did not recover consciousness and died four hours later.

The third (G14) had no records that could be deciphered until 15 minutes after valvotomy, when there was ventricular fibrillation that—in contrast with the previous patients—continued until his death which took place in the operating theatre.

The fourth (G73) had the usual short paroxysms of ventricular tachycardia during valvotomy. The rate then slowed and the patient collapsed with complexes that became wider and more variable in shape and rate (50–175): they were thought to be multifocal ventricular extrasystoles, not ventricular fibrillation. The S–T interval became more depressed and the patient died soon after returning to the ward without much further cardiographic change.

Ventricular fibrillation occurred also in two patients who did well. In the first (G29) there was clinical cardiac arrest after valvotomy, the longest asystole in the graphic records being 3 seconds in a period of slow irregular ventricular complexes with deep S–T III depression. After adrenalin and methedrine the rhythm quickened with more irregular complexes and changed to ventricular fibrillation which lasted 30 seconds. There was a return of sinus tachycardia with a more gradual return of normal QRS and S–T complexes: the patient made a good recovery.

In the second (G33) there were paroxysms of ventricular tachycardia during valvotomy and soon after this ventricular flutter and fibrillation, each for about 3 seconds; the rhythm quickly returned to the usual auricular fibrillation.

To sum up, three of the patients who died had ventricular flutter and fibrillation and the fourth had a slow multifocal ventricular rhythm. There were, however, two other patients with ventricular fibrillation who made a good recovery. In three of the five with ventricular fibrillation, it followed a period of asystole or A-V block. In the operations for mitral valvotomy the P–R interval often lengthened (once with dropped beats) but no higher grade of block than this was observed except in two of these patients (G7 and G15) who died.

During Angiocardiography. Although the effects of the injection of diodone—mainly depression of conduction and S–T changes—differ from those of operations for pulmonary or mitral stenosis, a few patients will be discussed briefly, to compare the cardiac mechanism. There were three patients who died after angiocardiography (Reynolds, 1953).

In one there were widened QRS complexes and S–T depression a minute after the injection of diodone. One minute later, the patient collapsed with increasing bundle branch block, followed by ventricular tachycardia, flutter, and fibrillation, which continued with a diminishing voltage until his death two hours later (Reynolds, 1953, Fig. 2).

In the second, a fall of blood pressure and the onset of bundle branch and partial A-V block occurred simultaneously four minutes after the injection and were followed by ventricular extrasystoles, singly and in short paroxysms, irregular sinus bradycardia, and then irregular ventricular tachycardia which persisted until cardiac massage was discontinued. Auricular and ventricular complexes could be recognized some time after the patient was thought to have died (Reynolds, 1953, Fig. 3).

In the third, there was partial A-V block and bundle branch block even before the injection: 8 minutes after it the patient collapsed with a slow ventricular rhythm. An auricular rhythm with a high grade of A-V block returned from time to time, but the rhythm and the ventricular complexes both became more abnormal and slower and continued after the patient was dead (Reynolds, 1953, Fig. 4).

Three of the patients having angiocardiography showed rather similar changes without causing any clinical anxiety. His Cases 7 and 8 developed severe S–T depression and bundle branch block but not for much more than a minute. Case 9 showed severe depression of A-V and intra-ventricular conduction.

Three others caused clinical anxiety but made a good recovery. One collapsed half an hour after the injection; no more than S–T depression had been observed, but no tracing was available at the time of her collapse. One showed partial A-V block with increasingly wide QRS complexes but the collapse did not come till 21 minutes later when there was complete bundle branch block. The third showed only slight changes until his sudden collapse six minutes after the injection, when there was partial A-V block which lasted for 20 minutes.

Here ventricular fibrillation was seen in one patient only and A-V block and bundle branch block were seen more often: in fact, depression of conduction seemed the most common danger.

Terminal Rhythms of the Heart

In this series the last record was nearly always a slow ventricular rhythm, with A-V dissociation if there was any evidence of auricular activity, whatever the heart's mechanism at the time the

patient was dying. Sometimes this could be seen fairly near the end (Cases 3 and 5) and sometimes right up to the end (Cases 2 and 4).

This was not true of the patient's dying during mitral valvotomy: in two of the four normal rhythm had returned, and the patients left the operating theatre but died about four hours later without recovering consciousness, and there are no records of this period. The other two died in the theatre, one with persistent ventricular fibrillation, the voltage gradually becoming lower; and the other, who had auricular fibrillation before operation, with little change other than widened QRS complexes and S–T depression.

Of the three patients who died during angiocardiography one had persistent ventricular fibrillation with complexes of progressively lower voltage. The other two had respectively a slow ventricular rhythm and an irregular ventricular tachycardia at the time they were thought to have died, but independent slow auricular and ventricular rhythms could be distinguished for a long time afterwards.

A good deal has been written about the electrical mechanism of the dying heart, since Robinson (1912) found that either the ventricle or the auricle might continue beating longest, that there was often A-V dissociation, and that electrocardiographic changes might continue for thirty-five minutes after clinical death, as in our records. Turner (1931), reviewing 65 cases and some of his own, found the mechanism very varied, but slowing of the heart was the most constant sign, occurring in 70 per cent: there might be nodal or idio-ventricular rhythms and sometimes terminal ventricular fibrillation. As the changes appeared only a few minutes before clinical death he did not think them likely to be of practical help. He often found changes in the ventricular complexes, a decrease of R and an increase of T with the two running together so that there was gross S–T elevation—a form that we have seen in some of these records. More recently Stroud and Feil (1948), found that slow abberrant ventricular complexes were common terminally, that standstill of the heart was the terminal mechanism in at least half of the patients, and that ventricular fibrillation was not as frequent as had sometimes been suggested. Harris (1948) in experimental results on dogs found that ventricular fibrillation occurred in about half the cases dying after coronary occlusion, but when anoxia was the factor, fibrillation was rare and changes of pacemaker and A-V block were the usual start of the terminal changes.

Summary

The electrocardiographic changes that have been seen with continuous recording during 70 direct operations for pulmonary stenosis, both valvular and infundibular, have been analysed and discussed. Patients with Fallot's tetralogy and those with pulmonary stenosis and a closed ventricular septum do not differ greatly in this respect, so have generally been discussed together.

Changes in the pacemaker, nodal rhythms, and A-V dissociation, with or without interference, are all very common; they are probably due to the anæsthetic rather than to the operation itself and are not of much significance.

There were as a rule some ventricular extrasystoles, but generally not many, though half the patients showed short paroxysms of ventricular tachycardia at some stage of the operation, rather more often when procaine was first injected into the myocardium and during the early stages of valvotomy or infundibular resection. They were not so common nor so long as the similar paroxysms observed during mitral valvotomy but, like these, they were often irregular in timing and sometimes multifocal in form.

Bundle branch block did not generally seem of serious significance and was much less common than during mitral valvotomy.

S–T depression with reciprocal elevation occurred during the injection of procaine into the ventricular muscle but passed off quickly. Direct electrograms showed how large this depression elevation was over the site of the injection though the reflection of it in the standard leads was much less. S–T depression was seen at other times also but did not seem of grave significance though in its extreme form it is probably due to anoxia or trauma to the heart. It had generally passed off before the operation ended.

Auricular fibrillation was not encountered. Ventricular fibrillation was seen in three patients; two of these died during the operation but the third recovered for ten days to succumb to the cerebral effects sustained when his circulation was ineffective. A-V block, complete or 2:1, occured in three patients, two of whom died.

The significance of these results has been discussed, and especially the changes found in the patients who died. They have been compared with the similar changes observed during operations for mitral valvotomy and during angiocardiography. The electrocardiographic patterns seen in the dying heart have been discussed briefly.

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